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Stimulation of Bone Growth Through Sports

A Radiologic Investigation of the Upper Extremities in Professional Tennis Players

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ABSTRACT

This contribution addresses the following questions: Does unilateral sports-specific strain affect the skeletal system of the athlete? Specifically, can any differences be found in longitudinal growth of the bones of the forearm and hand in professional tennis players between the stroke arm and the contralateral arm? An investigation was conducted involving 20 high-ranking professional tennis players (12 male and eight female players) between 13 and 26 years of age as well as 12 controls of the same age range. The radiologic examinations of the bones of the forearm and hand yielded an increase in density of bone substance and bone diameter as well as length in the stroke arm as compared with the contralateral arm. Whereas the first results confirm previous findings, the stimulation of longitudinal growth has never been reported. This change in bone structure and size can be attributed to two factors: mechanical stimulation and hyperemia of the constantly strained extremity. It may thus be regarded as a biopositive adaptation process.

Within the last 10 years, the high-ranking professional tennis players have become younger. This tendency applies to men as well as women players. Nowadays, players turn professional at the ages of 15 to 17. They start full-time practice at much earlier ages because of the technical complexity of tennis and the high degree of motor skills required. The best time to acquire these skills has been shown to be between ages 10 and 12 for girls and ages 10 and 13 for boys.[26] During this period, the differences between boys and girls in the development of their performance graphs are very small[27]; therefore, professional practice is begun during childhood. It is the responsibility of the physician specializing in sports medicine to direct the load of training to prevent injuries and overuse reactions. To fulfill this task, the physician has to know exactly how the athlete's growing body will react to high loads of practice.

Functional adaptation of bone, cartilage, and muscles has been described by Feldmeier.[10] He found an increase of bone thickness, bigger bone ledges at the insertion of muscles and tendons, and a concentration of bone structure. Wurster et al.[39] referred to the interdependency of blood estradiol and bone density in female track and field athletes; however, an influence of sports-specific strain on the longitudinal bone growth has not been reported.

Growth and development of the skeleton

Skeletal growth and development are affected by several factors.[26] Any growth is caused by cell division. Reduplication is determined by an intracellular genetic code. Another important factor is nutrition; malnutrition will result in a retardation of growth. Third, the hormone-related factors, especially the feedback regulation of calcitonin and parathormone, play important roles in bone development. Estradiol reduces bone resorption[39]; somatotrophic hormone supports growth in general, i.e., its effect is not limited to bones. The final body height at the end of growth is determined by the genetic code[9] but can be affected by the factors mentioned above. Matthiass[26] found systemic as well as local regulatory mechanisms of skeletal growth. Bone growth is characterized by a constant remodeling in which gain and loss are not balanced. Systemic regulations

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happen through hormonal control loops. Harris and Heaney[14] reported the interaction of parathormone and calcitonin. Kaye et al.[20] found a stimulation of bone cell proliferation caused by estradiol and testosterone. Local regulation is achieved by biomechanical and biophysical means.[3] The effect of local influence is demonstrated by the different growth patterns of the proximal and distal epiphyseal plates of the extremities. It has been shown that the distal epiphyseal plate has a larger part in longitudinal growth than the proximal one for the bones of the forearm[9] (Fig. 1). At the distal end of the forearm, the ulnar epiphyseal plate contributes 81%, and the radial epiphyseal plate 75% to the total growth. This is the reason for the late ossification that takes place only at the end of the 2nd decade.[13] Thus, the forearm provides an excellent area for investigating the influence of external factors like high strain on bone growth.

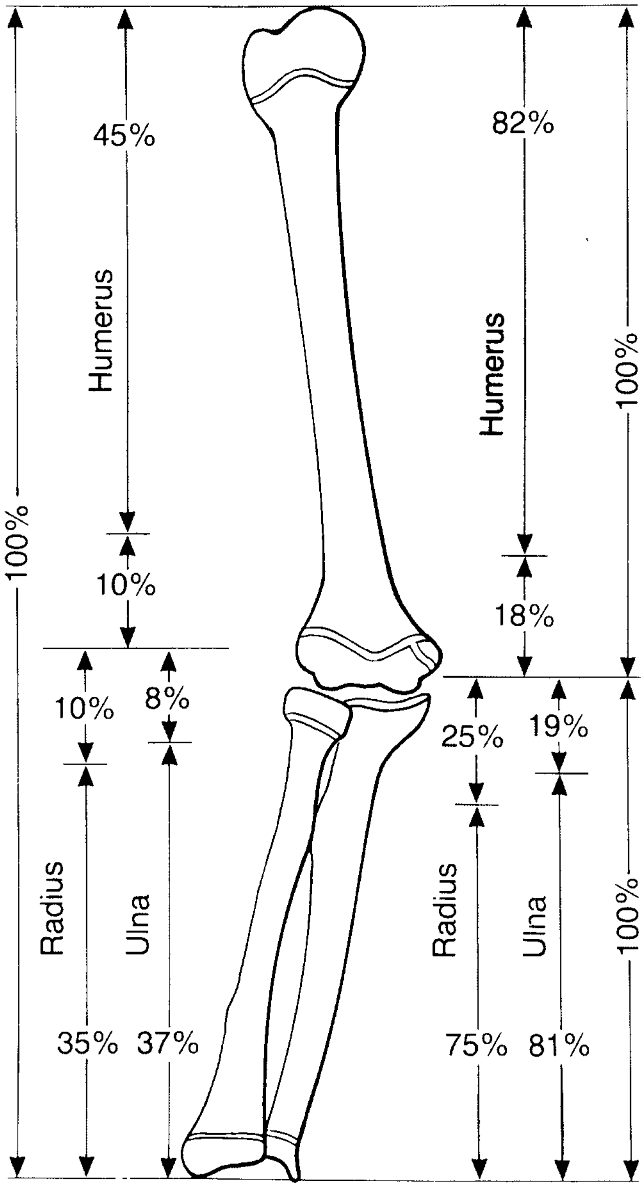

Figure 1. Contribution of the different epiphyseal plates to the total growth of the arm. (Used with permission from Exner GU: *Normwerte der Kinderorthopädie.* Stuttgart, Thieme-Verlag, 1990.)

Skeletal reactions to sports-specific strain

It is well known that physical exercise causes hypertrophy in the muscles involved. If there is a different degree of strain on the two extremities, the extent of muscular hypertrophy will also differ.[5] Continuous mechanical stress results in adaptation consisting of an increase in cortical thickness and growth of the bone ledges at the insertion of muscles and tendons.[36] Ilizarov[18] described a "tension-stress-effect" leading to growth stimulation and the formation of new bone cells in a study on the tibial bones of dogs. Hinsenkamp and Rooze[16] reported changes in morphology and metabolism in different parts of the bone after stimulation in an electromagnetic field. Hinsenkamp[15] found diaphyseal lengthening in the legs of fetal mice after stimulation as well as a positive effect on bone growth stimulation in the treatment of pseudarthrosis. In a study involving the effect of sports-specific strain, Jones et al.[19] found a significant hypertrophy in the humerus of the stroke arm in tennis players. Buskirk et al.,[5] Krahl,[23] and Krahl et al.[24] reported similar results for the forearm. The bones of the hand and the elbows of tennis-playing children are also known to exhibit adaptation to high-stress loads.[8,17,23,24]

In addition to those indications of biopositive bone adaptation, there are also pathologic reactions to overuse. Murakami[29] reported a stress fracture of the second metacarpal in the stroke arm of a competitive tennis player. As a result of repeated microlesions due to excessive practice loads, arthrosis becomes more and more frequent.[12]

Stimulation of the epiphyseal plate

Skeletal growth takes place in the epiphyseal plate[26] by ossification of cartilage. From a clinical point of view, we distinguish between two parts of the epiphyseal plate: the epiphyseal part with the potential to proliferate and the metaphyseal part without this potential.[37] The former consists primarily of matrix components, the latter of cellular components. These are more susceptible to shearing and bending forces, which is why slipping of the epiphyseal plate takes place in this area. Thus, the germinative layer has better protection in the epiphyseal part, and longitudinal growth is not impeded in proper epiphysiolysis.[37]

Stimulation of the epiphyseal plate can be achieved by different means. For one thing, an increased vascularization results in an increase in longitudinal growth provided it takes place during the phase of general growth.[37] Reactive hyperemia is a well-known consequence of bone fractures.[11,30,32] If the trauma occurs during a period of premature rest, the increase in length is temporary, whereas during epiphyseal fusion this process may be accelerated and may, in turn, result in a permanent shortening. As Rodegerdts[33] demonstrated, osteotomies may also stimulate epiphyseal growth. Finally, any diseases accompanied by bone hyperemia such as tumors, osteomyelitis, or Klippel-Trenaunay syndrome may cause additional growth.[26]

There is no detailed knowledge as yet on the influence of mechanical factors on the behavior of the epiphyseal plate. Although Basset[1] was able to show piezoelectricity in eccentric strain on the bone, it is still not clear whether this results in an accelerated cell flow in the germinative zone that is commonly regarded as the receptor of local or systemic stimulation.

MATERIALS AND METHODS

Between July 1986 and October 1991, we examined a total of 20 nationally and internationally high-ranking tennis professionals (12 male and eight female players). They were between 13 and 26 years of age (mean, 20.1) at the time of the examination. All of them had histories of at least 5 years of training and competition. They had started serious practice at no later than age 11. The daily practicing time had to be a minimum of 2 hours. Eighteen of the subjects were right-handed; two were left-handed.

We examined the effect of constant strain caused by intensive training and competition on the longitudinal growth of the bones in the forearm and hand by comparing radiographic measurements of the stroke arm with those of the contralateral arm of the same subject. In addition, the same measurements were performed in a control group of the same age range (five female and seven male subjects; mean age, 23.1 years; 3 were left- and 9 right-handed). None of those included in the control group did any unilateral manual labor or suffered from any severe systemic illness or bone lesions. Each subject or control was examined once.

The following parameters were investigated:

1. Total length of the ulna (olecranon to styloid process of the ulna),
2. Total length of the second metacarpal,
3. Diameter of the ulna 10 cm proximal to the styloid process, and
4. Diameter of the second metacarpal at a distance of 3 cm from the distal end.

Radiographs of all subjects were made by using a Philips BT-S4 machine (Eindhoven, Holland) on AGFA Crurix RP1L (AGFA Co., Köln, Germany) with a film focus distance of 100 cm (44 kV/22 mA/sec). Interindividual variation was minimized by having this standardized procedure implemented by the same operator and instrument. The interpretation of each film was done by the same investigator (UM) to minimize further variations.

Each hand or lower arm was radiographed individually perpendicular to the film to avoid positional magnification and distortion. The limb was positioned at 90° of pronation and 45° of elbow flexion, and was controlled during the radiographic procedure by the same investigator.

There was no magnification induced by soft tissue thickness or by possible side differences in muscle thickness, as Eichel[8] has shown.

RESULTS

The visual inspection of the radiographs suggests an obvious difference in bone structure between the stroke arm and the contralateral arm for the tennis players. This concerns first of all the bone thickness already observed as early as 1956 by Buskirk et al.[5]

The diameter of the ulna was larger in the stroke arm of every athlete than in the nondominant arm with an average of 2.1 mm (range, 1.0 to 4.4). Probability, calculated using the t-test, was $P < 0.01$ (Table 1). There was no such lateral difference in the control group (Table 2; $P < 0.001$).

In addition to this well-known effect, however, we observed a highly significant difference in ulnar length between the two arms in the tennis players (Fig. 2). Differences ranged from 0.2 to 1.3 cm (mean, 0.8; Table 3). Probability, calculated using the t-test, was $P < 0.01$. The difference was observed in male as well as female subjects. Again, there was no such lateral difference in the control group (Table 4, Fig. 3; $P < 0.001$).

Regarding the second metacarpal, tennis players exhibit an increase in bone density as well as cortical thickness in the stroke arm as compared with the collateral arm (Fig. 4). The average difference in diameter was 1.0 mm with a minimum difference of 0.1 and a maximum of 2.1 mm in favor of the dominant arm (Table 5). In addition, the athletes showed an average lengthening of the second metacarpal in the stroke arm of 2.7 mm (range, 0.0 to 4.7), again highly significant (Table 6). Probability, calculated using the t-test, was $P < 0.01$. Once again, there were no differences between men and women. The control group showed no lateralization (Tables 7 and 8; Fig. 5; $P < 0.001$).

The differences in length between the stroke arm and the contralateral arm thus amount to 3% for the ulna and 3.7% for the second metacarpal. Statistical evaluation shows no significant correlation between the length differences of

TABLE 1
Diameter of the ulna in professional tennis players (10 cm proximal to the styloid process, in millimeters)

Age (years)	Sex	Handedness	Stroke arm	Contralateral arm	Difference
24	M	Right	15.1	13.2	1.9
14	M	Left	13.3	11.0	2.3
26	M	Right	15.9	13.1	2.8
13	M	Right	11.6	9.9	1.7
20	M	Right	15.3	13.2	2.1
26	M	Right	15.0	13.9	1.1
16	M	Right	12.2	11.2	1.0
24	M	Right	12.9	10.2	2.7
21	M	Right	13.1	10.9	2.2
22	M	Right	15.8	11.4	4.4
26	M	Right	11.8	10.0	1.8
19	M	Right	14.6	13.0	1.6
20	F	Right	13.1	10.5	2.6
23	F	Right	11.9	10.3	1.6
19	F	Right	10.9	9.4	1.5
15	F	Right	11.9	9.3	1.7
25	F	Right	12.5	10.0	2.5
20	F	Right	12.3	9.4	2.9
14	F	Left	12.3	11.0	1.9
14	F	Right	11.0	9.6	1.4
20.1[a]			13.1[a]	11.0[a]	2.1[a]

[a] Mean.

TABLE 2
Diameter of the ulna (10 cm proximal to the styloid process, in millimeters) control group

Age (years)	Sex	Handedness	Dominant arm	Nondominant arm	Difference
27	M	Left	13.4	13.4	0.0
20	M	Right	12.5	12.5	0.0
25	M	Right	11.7	11.7	0.0
18	M	Left	14.1	14.2	-0.1
15	M	Right	12.4	12.4	0.0
28	M	Right	14.0	14.0	0.0
19	M	Right	13.3	13.1	0.2
14	F	Right	10.6	10.7	-0.1
24	F	Right	11.5	11.5	0.0
22	F	Right	12.7	12.6	0.1
16	F	Right	10.9	10.9	0.0
27	F	Left	12.3	12.2	0.1
23.1[a]			12.45[a]	12.43[a]	0.02[a]

[a] Mean.

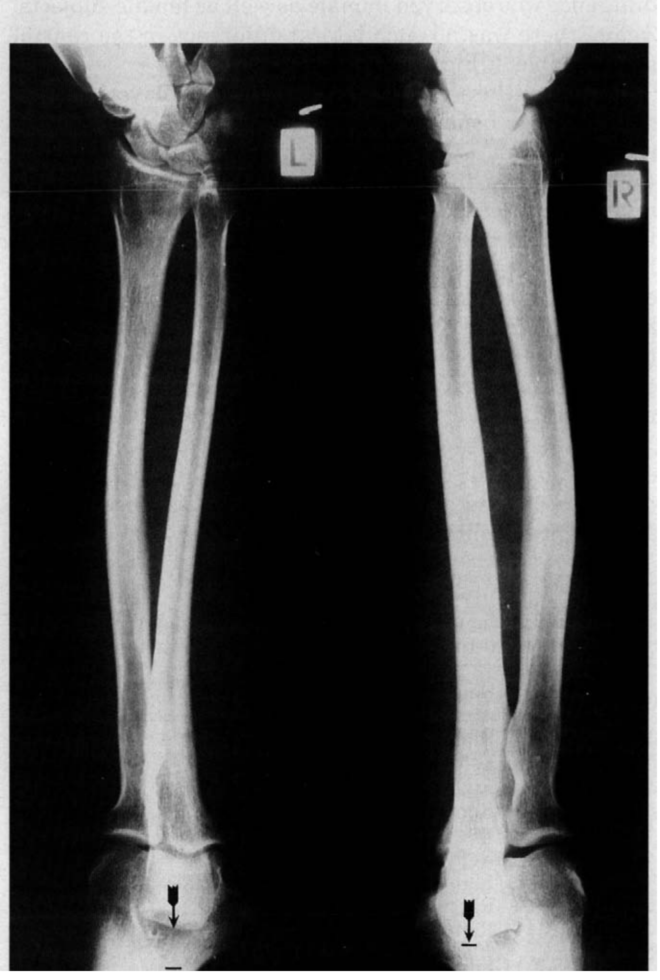

Figure 2. The forearms of a tennis professional (right-hander) with a hypertrophy of the right arm (widening of 4.4 mm, lengthening of 13 mm).

the ulna or second metacarpal and the age of the athlete at the time of the examination. Statistical evaluation concerning the influence of the daily amount of training time or training intensity on stimulation of bone growth was not possible because of the lack of exact data.

TABLE 3
Total length of the ulna in professional tennis players (olecranon to styloid process, in millimeters)

Stroke arm	Contralateral arm	Difference
281	275	6
246	241	5
300	288	2
256	252	4
304	296	8
292	286	6
282	272	10
329	323	6
314	304	10
301	288	13
287	278	9
280	270	10
271	260	11
274	264	10
248	244	4
246	237	9
272	262	10
278	269	9
264	254	10
250	243	7
278[a]	270[a]	8[a]

[a] Mean.

TABLE 4
Total length of the ulna (olecranon to styloid process, in millimeters) control group

Dominant arm	Nondominant arm	Difference
273	272	1
255	255	0
304	304	-1
277	277	0
258	258	0
289	288	1
265	265	0
248	248	0
254	254	0
270	270	0
269	268	1
281	281	0
270[a]	270[a]	0.17[a]

[a] Mean.

DISCUSSION

Sport-specific unilateral strain on the growing skeleton of the professional tennis player results in impressive adaptive processes. A different development, especially of the forearms, can clearly be seen in the photograph of a former top-ten tennis player (Fig. 6).

One-sided bone hypertrophies were recorded in several sports.[5,7,8,17,23,24] Jones et al.[19] examined the cortical thickness of the humerus in tennis players. Their subjects showed a significant widening of the corticalis in the stroke arm as compared with the contralateral arm of 34.9% for the men and 28.4% for the women.

First, there is an increase in bone width in the stroke arm. At the same time, an increase in density can be observed. The question of whether these increases are caused by bone hypertrophy or bone hyperplasia has to remain

Figure 3. The forearms of a right-hander from the control group without significant differences in bone development between left and right arms.

open. Further biochemical or densitometric investigations as described by Rüegsegger[34] are required.

Unilateral bone hypertrophy has been described for different sports: Dahlen and Olsson[7] described a 20% increase of bone trabeculae in long-distance runners as compared with a control group not actively involved in sports. Buskirk et al.,[5] Eichel,[8] and Krahl[23] reported alterations of bone structure in forearms and hands of tennis players.

Differences in length—as observed in the present study—have not been described before. On the contrary, Mocellin[28] stated that sports-specific strain constituted neither a positive nor negative effect on longitudinal bone growth. We believe that this theory can no longer be upheld. Because we did not detect any effect on bone width or length in the control group, the findings described above for the tennis players cannot be attributed to handedness but must be interpreted as consequences of sports-induced strain. As expected,[19] no differences between male and female athletes were observed.

The question of how to interpret the lengthening of the ulna and second metacarpal remains. According to Wolff,[38] all bone growth is determined by functional forces. The potential of growth is directly proportional to local stress effects. The bone will grow axially as long as stress is effected on the full width of the epiphyseal plate.[25]

In tennis there is an enormous strain on the forearm and hand of the dominant arm.[22] Any stroke will transmit

Figure 4. Hands of a tennis professional (right-hander) with significant changes of the right as compared with the left second metacarpals (widening of 1.6, lengthening of 4.1 mm).

TABLE 5
Diameter of the second metacarpal in professional tennis players (3 cm from the distal end, in millimeters)

Stroke arm	Contralateral arm	Difference
10.5	8.4	**2.1**
9.0	8.5	**0.5**
10.4	9.4	**0.5**
9.5	8.8	**0.7**
10.2	9.3	**0.9**
10.4	9.7	**0.7**
9.4	9.3	**0.1**
9.9	9.7	**0.2**
9.3	8.5	**0.8**
12.3	10.7	**1.6**
9.2	7.6	**1.6**
12.1	10.4	**1.7**
9.8	8.9	**1.8**
11.4	10.1	**1.3**
8.1	7.7	**0.4**
8.7	7.7	**1.0**
8.7	7.5	**1.2**
8.9	8.2	**0.7**
8.8	8.3	**0.5**
9.4	8.6	**0.8**
9.8[a]	8.9[a]	0.9[a]

[a] Mean.

TABLE 6
Total length of the second metacarpal in professional tennis players (in millimeters)

Stroke arm	Contralateral arm	Difference
75.8	71.7	4.1
66.7	64.3	2.4
82.2	78.7	3.5
75.9	74.2	1.7
66.7	64.9	1.8
77.8	75.4	2.4
75.1	72.3	2.8
81.2	77.0	4.2
82.3	79.4	2.9
84.0	79.9	4.1
74.5	71.7	2.8
77.0	73.3	3.7
72.8	69.6	3.2
79.1	77.4	1.7
70.7	70.7	0.0
68.3	68.3	0.0
71.2	70.3	0.9
78.9	74.8	4.1
73.0	71.1	1.9
62.0	57.3	4.7
74.8[a]	72.1[a]	2.7[a]

[a] Mean.

TABLE 7
Diameter of the second metacarpal (3 cm from the distal end, in millimeters) control group

Dominant arm	Nondominant arm	Difference
9.1	9.1	0.0
8.5	8.5	0.0
9.3	9.3	0.0
9.7	9.7	0.0
8.4	8.4	0.0
10.0	10.0	0.0
8.6	8.6	0.0
8.2	8.2	0.0
8.7	8.7	0.0
9.0	9.0	0.0
7.9	7.9	0.0
8.1	8.1	0.0
8.8[a]	8.8[a]	0.0[a]

[a] Mean.

TABLE 8
Total length of second metacarpal (in millimeters) control group

Dominant arm	Nondominant arm	Difference
72	72	0
69	69	0
77	77	0
81	81	0
70	70	0
78	78	0
76	76	0
64	64	0
69	69	0
73	73	0
72	72	0
74	74	0
73[a]	73[a]	0[a]

[a] Mean.

Figure 5. Hands of a right-hander out of the control group without changes of the metacarpals.

mechanical stimuli (vibrations) from the racket to the hand. Because the absorption of these vibrations by the racket is very slow, most of them have to be absorbed by the hand, as demonstrated by Brody.[4] Thus, there is a very strong mechanical component that may very well result in a stimulation of the epiphyseal plate by way of piezoelectric effects.[35]

The second factor seems to be a temporary hyperemia of the muscular system of the dominant arm in tennis players that is induced by sports-specific strain.[6,21] An increase of length growth from pathologic hyperemia in osteomyelitis or Klippel-Trenaunay syndrome is well known.

In summary, the unilateral lengthening can be regarded as a result of the specific strain induced by tennis practice and as a biopositive adaptive reaction. The mechanical strain and hyperemia seem to be able to stimulate the germinative cell layer of the epiphyseal plate in some way. However, the exact size, direction, and duration of the effective forces and the exact increase in vascularization have not as yet been determined because of a lack of a model of biomechanical force. Our observation of an increase in bone diameter and density confirms the findings of other authors.[5,6,8,17,23,24]

The question of whether the changes observed correlate with the intensity of practice strain cannot be provided with a definite answer by this study because, beyond the fact that we only included top national and international professional tennis players, we did not record the exact number of practice hours or the amount of practice intensity over the years. However, a minimum of 10 hours per week (average, 20 hours) was adopted as a criterion for the selection of the subjects.

This study is the first reported observation of intraindividual variation of bone length of the ulna and second metacarpal in professional tennis players' stroke and collateral arms. The small but significant growth was consistent across all subjects in the study and was absent in the control

Figure 6. The forearms of a former top-ten tennis professional (left-hander).

group with corresponding age and sex. Although the clinical significance of these data has yet to be fully understood, it is suggested that the mechanical stimulation of the stroke arm and associated hyperemia are the causative factors for this growth. Future biomechanical and hematologic studies should be designed to further explore this possibility both in healthy athletic populations and in populations that could benefit from increasing bone growth.

Of course, certain reactions like stress fractures and other overuse reactions will always have to be watched for.[2,29,31]

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